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Terms	Documents
ced-9 and l2	0

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L4	ced-9 and l2	0	L4
L3	l1 and L2	0	L3
L2	n3400 or n3407 or n3377	19	L2
L1	ced-9 near5 mutat$	18	L1

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Search Results - Record(s) 1 through 18 of 18 returned.

☐ 1. 20030023994 . 22 Jun 01. 30 Jan 03. Assay for toxin induced neuronal degeneration and viability in C. elegans. Blakely, Randy D., et al. 800/9; 800/13 A01K067/033 G01N033/00.

☐ 2. 20030023061 . 14 May 02. 30 Jan 03. Family of genes encoding apoptosis-related peptides, peptides encoded thereby and methods of use thereof. Umansky, Samuil, et al. 536/23.2; 435/183 435/320.1 435/325 435/69.1 435/7.23 G01N033/574 C07H021/04 C12N009/00 C12P021/02 C12N005/06.

☐ 3. 20020136714 . 22 Jun 01. 26 Sep 02. Relatedness of human interleukin-1beta convertase gene to a C. elegans cell death gene, inhibitory portions of these genes and uses therefor. Horvitz, H. Robert, et al. 424/94.63; 435/226 435/320.1 435/325 435/69.1 A61K038/48 C12N009/64 C12P021/02 C12N005/06.

☐ 4. 20020007496 . 16 May 01. 17 Jan 02. Methods for identifying novel therapeutics and diagnostics in the p53 pathway. Rothman, Joel H., et al. 800/8; 536/23.2 A01K067/033 C07H021/04.

☐ 5. 6465617 . 20 Jan 99; 15 Oct 02. Identification and characterization of a gene which protects cells from programmed cell death and uses therefor. Horvitz; H. Robert, et al. 530/350; 536/23.1 536/23.5. C07K001/00 C07K014/00 C07K017/00 C07H021/02 C07H021/04.

☐ 6. 6433155 . 24 Sep 97; 13 Aug 02. Family of genes encoding apoptosis-related peptides, peptides encoded thereby and methods of use thereof. Umansky; Samuil, et al. 536/23.5; 435/320.1 435/325 435/69.1 536/23.1 536/24.3 536/24.31 536/24.33. C07H021/04 C07H021/02 C12P021/06 C12N015/00.

☐ 7. 6312947 . 20 Jan 99; 06 Nov 01. Identification and characterization of a gene which protects cells from programmed cell death and uses therefor. Horvitz; H. Robert, et al. 435/320.1; 435/325 435/410 435/455 536/23.1 536/23.5. C07H021/02 C07H021/04 C12N015/00 C12N015/63 C12N005/00.

☐ 8. 6221615 . 25 Jan 99; 24 Apr 01. Peptides and compositions which modulate apoptosis. Chittenden; Thomas D., et al. 435/7.1; 530/324 530/327. G01N033/53 C07K016/00.

☐ 9. 6184202 . 11 Sep 97; 06 Feb 01. Cell death regulators. Korsmeyer; Stanley J.. 514/12; 514/2. A61K038/00.

☐ 10. 6087160 . 04 Jan 95; 11 Jul 00. Programmed cell death genes and proteins. Yuan; Junying, et al. 435/320.1; 435/219 435/226 435/69.1 536/23.2 536/23.5. C12N015/63.

☐ 11. 6083735 . 10 Jun 94; 04 Jul 00. Programmed cell death genes and proteins. Yuan; Junying, et al. 435/226; 424/94.67 435/252.3 435/320.1 530/350 536/23.2 536/23.5. C12N009/64.

☐ 12. 5962301 . 24 Feb 95; 05 Oct 99. Relatedness of human interleukin-1.beta. convertase gene to a C. elegans cell death gene, inhibitory portions of these genes and uses therefor. Horvitz; H. Robert, et

al. 435/226; 435/219. C12N009/64 C12N009/50.

☐ 13. 5863795 . 08 Aug 97; 26 Jan 99. Nucleic acids that encode peptides which modulate apoptosis. Chittenden; Thomas D., et al. 435/325; 435/243 435/320.1 435/410 536/23.5 536/24.31. C12N005/10 C12N001/00 C12N015/12 C12N015/63.

☐ 14. 5856171 . 10 Nov 94; 05 Jan 99. Cell death regulators. Korsmeyer; Stanley J.. 435/254.2; 435/252.3 435/6 435/810 530/324 530/350 530/388.1 530/389.1 530/827 536/23.5. C12N001/15 C07H021/04 C07K014/435.

☐ 15. 5700638 . 25 May 94; 23 Dec 97. Cell death regulator. Korsmeyer; Stanley J.. 435/6; 435/477 435/69.1 435/7.1 435/7.2 435/7.21 435/7.31 435/7.8 436/501 530/350. C12Q001/68 G01N033/68 G01N033/53 C12P021/02 C12N005/02 C12N005/12 C07K014/475.

☐ 16. 5656725 . 12 May 95; 12 Aug 97. Peptides and compositions which modulate apoptosis. Chittenden; Thomas D., et al. 530/324; 530/325 530/326 530/327 530/328 530/329 530/330. C07K007/06 C07K007/08 C07K014/47.

☐ 17. 5614397 . 22 Feb 94; 25 Mar 97. Method and compositions for modulating lifespan of hematolymphoid cells. Weissman; Irving, et al. 435/458; 435/325 435/355. C12N015/85 C12N005/10.

☐ 18. 5196333 . 30 May 90; 23 Mar 93. DNA sequences involved in neuronal degeneration, multicellular organisms containing same and uses thereof. Chalfie; Marin, et al. 435/369; 435/29 435/69.1 435/70.3 536/23.5. C12N005/00 C07H015/12 C12P021/06 C12Q001/68.

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Terms	Documents
ced-9 near5 mutat$	18

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=> d his

(FILE 'HOME' ENTERED AT 16:40:30 ON 03 MAR 2003)

FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 16:40:57 ON 03 MAR 2003

L1 470 S CED-9
L2 35 S MUTAT?(5A)L1
L3 12 DUP REM L2 (23 DUPLICATES REMOVED)

=> d bib ab 1-12 l3

L3 ANSWER 1 OF 12 MEDLINE DUPLICATE 1
AN 2000472680 MEDLINE
DN 20418097 PubMed ID: 10846174
TI Disruption of the CED-9.CED-4 complex by EGL-1 is a critical step for programmed cell death in Caenorhabditis elegans.
AU del Peso L; Gonzalez V M; Inohara N; Ellis R E; Nunez G
CS Department of Pathology and Comprehensive Cancer Center and the The University of Michigan, Ann Arbor, Michigan 48109, USA.
NC CA-64556 (NCI)
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (2000 Sep 1) 275 (35) 27205-11.
Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200010
ED Entered STN: 20001012
Last Updated on STN: 20001012
Entered Medline: 20001003
AB In the nematode Caenorhabditis elegans, the apoptotic machinery is composed of four basic elements: the caspase CED-3, the Apaf-1 homologue CED-4, and the Bcl-2 family members CED-9 and EGL-1. The **ced-9**(n1950) gain-of-function **mutation** prevents most, if not all, somatic cell deaths in C. elegans. It encodes a CED-9 protein with a glycine-to-glutamate substitution at position 169, which is located within the highly conserved Bcl-2 homology 1 domain. We performed biochemical analyses with the CED-9G169E protein to gain insight into the mechanism of programmed cell death. We find that CED-9G169E retains the ability to bind both EGL-1 and CED-4, although its affinity for EGL-1 is reduced. In contrast to the behavior of wild-type CED-9, the interaction between CED-9G169E and CED-4 is not disrupted by expression of EGL-1. Furthermore, CED-4 and CED-9G169E co-localizes with EGL-1 to the mitochondria in mammalian cells, and expression of EGL-1 does not induce translocation of CED-4 to the cytosol. Finally, the ability of EGL-1 to promote apoptosis is impaired by the replacement of wild-type CED-9 with CED-9G169E, and this effect is correlated with the inability of EGL-1 to induce the displacement of CED-4 from the CED-9.CED-4 complex. These studies suggest that the release of CED-4 from the CED-9.CED-4 complex is a necessary step for induction of programmed cell death in C. elegans.

L3 ANSWER 2 OF 12 MEDLINE DUPLICATE 2
AN 2001038980 MEDLINE
DN 20504438 PubMed ID: 11027303
TI Demonstration of the in vivo interaction of key cell death regulators by structure-based design of second-site suppressors.
AU Parrish J; Metters H; Chen L; Xue D
CS Department of Molecular, Cellular, and Developmental Biology, University of Colorado, Boulder, CO 80309, USA.
SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (2000 Oct 24) 97 (22) 11916-21.
Journal code: 7505876. ISSN: 0027-8424.

CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200011
ED Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001128
AB Demonstrating in vivo interaction of two important biomolecules and the relevance of the interaction to a biological process have been difficult issues in biomedical research. Here, we report the use of a homology modeling approach to establish the significance of protein interactions in governing the activation of programmed cell death in Caenorhabditis elegans. A protein interaction cascade has been postulated to mediate activation of cell death in nematodes, in which the BH3-domain-containing (Bcl-2 homology region 3) protein EGL-1 binds the cell-death inhibitor CED-9 and induces release of the death-activating protein CED-4 from inhibitory CED-4/CED-9 complexes. We show here that an unusual gain-of-function **mutation** in **ced-9** (substitution of glycine 169 to glutamate) that results in potent inhibition of most nematode cell deaths impairs the binding of EGL-1 to CED-9 and EGL-1-induced release of CED-4 from CED-4/CED-9 complexes. Based on a modeled EGL-1/CED-9 complex structure, we generated second-site compensatory mutations in EGL-1 that partially restore the binding of EGL-1 to CED-9(G169E) and EGL-1-induced release of CED-4 from CED-4/**CED-9**(G169E) complexes. Importantly, these **mutations** also significantly suppress the death-protective activity of CED-9(G169E) in vivo. These results establish that direct physical interaction between EGL-1 and CED-9 is essential for the release of CED-4 and the activation of cell death. The structure-based design of second-site suppressors via homology modeling should be widely applicable for probing important molecular interactions that are implicated in fundamental biological processes.

L3 ANSWER 3 OF 12 MEDLINE DUPLICATE 3
AN 2000156769 MEDLINE
DN 20156769 PubMed ID: 10688797
TI Translocation of C. elegans CED-4 to nuclear membranes during programmed cell death.
AU Chen F; Hersh B M; Conradt B; Zhou Z; Riemer D; Gruenbaum Y; Horvitz H R
CS Howard Hughes Medical Institute, Department of Biology, 68-425, Massachusetts Institute of Technology, Cambridge, MA 02139, USA.
SO SCIENCE, (2000 Feb 25) 287 (5457) 1485-9.
Journal code: 0404511. ISSN: 0036-8075.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200003
ED Entered STN: 20000327
Last Updated on STN: 20000327
Entered Medline: 20000313
AB The Caenorhabditis elegans Bcl-2-like protein CED-9 prevents programmed cell death by antagonizing the Apaf-1-like cell-death activator CED-4. Endogenous CED-9 and CED-4 proteins localized to mitochondria in wild-type embryos, in which most cells survive. By contrast, in embryos in which cells had been induced to die, CED-4 assumed a perinuclear localization. CED-4 translocation induced by the cell-death activator EGL-1 was blocked by a gain-of-function **mutation** in **ced-9** but was not dependent on ced-3 function, suggesting that CED-4 translocation precedes caspase activation and the execution phase of programmed cell death. Thus, a change in the subcellular localization of CED-4 may drive programmed cell death.

L3 ANSWER 4 OF 12 CAPLUS COPYRIGHT 2003 ACS
AN 1999:811447 CAPLUS
DN 132:32920
TI Detection of apoptotic cells in living nematodes by staining with vital dyes
IN Hengartner, Michael O.; Gartner, Anton; Milstein, Stuart
PA Cold Spring Harbor Laboratory, USA
SO PCT Int. Appl., 50 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9966323	A1	19991223	WO 1999-US13650	19990618
	W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	CA 2335549	AA	19991223	CA 1999-2335549	19990618
	AU 9945731	A1	20000105	AU 1999-45731	19990618
	AU 746694	B2	20020502		
	EP 1088226	A1	20010404	EP 1999-928736	19990618
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	JP 2002518674	T2	20020625	JP 2000-555091	19990618
PRAI	US 1998-90057P	P	19980619		
	WO 1999-US13650	W	19990618		

AB The present invention relates to methods and kits for detecting apoptotic cells in a live nematode by exposing the live nematode to a vital dye that stains apoptotic cells, and detecting the apoptotic cells. Applications of this assay include methods of identifying agents, conditions or genes that modulate apoptosis. The present invention also includes methods of screening for mutated nematodes that exhibit altered apoptotic cell death. Caenorhabditis elegans were stained with Acridine Orange. Mutant nematodes were made and studied.
RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 5 OF 12 MEDLINE DUPLICATE 4
AN 1998387925 MEDLINE
DN 98387925 PubMed ID: 9721101
TI Essential role of CED-4 oligomerization in CED-3 activation and apoptosis.
CM Comment in: Science. 1998 Aug 28;281(5381):1298-9
AU Yang X; Chang H Y; Baltimore D
CS Department of Biology, Massachusetts Institute of Technology, Cambridge, MA 02139, USA.
NC CA51462 (NCI)
SO SCIENCE, (1998 Aug 28) 281 (5381) 1355-7.
Journal code: 0404511. ISSN: 0036-8075.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199809
ED Entered STN: 19980917
Last Updated on STN: 20000303
Entered Medline: 19980909
AB Control of the activation of apoptosis is important both in development

and in protection against cancer. In the classic genetic model Caenorhabditis elegans, the pro-apoptotic protein CED-4 activates the CED-3 caspase and is inhibited by the Bcl-2-like protein CED-9. Both processes are mediated by protein-protein interaction. Facilitating the proximity of CED-3 zymogen molecules was found to induce caspase activation and cell death. CED-4 protein oligomerized in cells and in vitro. This oligomerization induced CED-3 proximity and competed with CED-4:**CED-9** interaction. **Mutations** that abolished CED-4 oligomerization inactivated its ability to activate CED-3. Thus, the mechanism of control is that CED-3 in CED-3:CED-4 complexes is activated by CED-4 oligomerization, which is inhibited by binding of CED-9 to CED-4.

L3 ANSWER 6 OF 12 CAPLUS COPYRIGHT 2003 ACS
AN 1998:324569 CAPLUS
DN 129:91808
TI Mutational analysis of Caenorhabditis elegans CED-4
AU Seshagiri, Somasekar; Chang, Wen-teh; Miller, Lois K.
CS Department of Entomology, The University of Georgia, Athens, GA, 30602, USA
SO FEBS Letters (1998), 428(1,2), 71-74
CODEN: FEBLAL; ISSN: 0014-5793
PB Elsevier Science B.V.
DT Journal
LA English
AB Much of our knowledge concerning the genetics that regulate cell death has come from the studies of cell death during the development of the nematode Caenorhabditis elegans. Of the 14 genes identified as components of nematode cell death pathways, two genes, ced-3 and ced-4, are required to promote cell death and a third, ced-9, blocks cell death. Recent studies show CED-4 to be an activator of CED-3 and CED-9 to be an inhibitor of CED-4. Two published sequence alignments suggest that CED-4 contains a death effector domain (DED), a protein sequence motif present in other death signaling proteins like Fadd and Flice; one study suggests a DED sequence similarity near the N-terminus while the other found sequence similarity near the C-terminus of CED-4. Using mutational anal. we have tested the functional significance of the conserved residues found within the putative DEDs of CED-4. Mutations in two conserved residues within the putative N-terminal DED of CED-4 affected its function, while mutations in the conserved residues within the putative C-terminal DED had no effect on CED-4 function. Our results do not support the presence of a DED in the C-terminus of CED-4 and suggest a potential role for the N-terminus in CED-4 function, possibly as a DED or as a CARD (caspase recruitment domain). We also found that CED-9 assocd. with all the CED-4 mutants and inhibited the activity of all the active-CED-4 mutants.
RE.CNT 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 7 OF 12 MEDLINE DUPLICATE 5
AN 97177114 MEDLINE
DN 97177114 PubMed ID: 9024666
TI Interaction between the C. elegans cell-death regulators CED-9 and CED-4.
AU Spector M S; Desnoyers S; Hoeppner D J; Hengartner M O
CS Cold Spring Harbor Laboratory, New York 11724, USA.
SO NATURE, (1997 Feb 13) 385 (6617) 653-6.
Journal code: 0410462. ISSN: 0028-0836.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199703
ED Entered STN: 19970313
Last Updated on STN: 19970313
Entered Medline: 19970303

AB Programmed cell death (apoptosis) is an evolutionarily conserved process used by multicellular organisms to eliminate cells that are not needed or are potentially detrimental to the organism. Members of the Bcl-2 family of mammalian proteins are intimately involved in the regulation of apoptosis, but, their precise mechanism of action remains unresolved. In Caenorhabditis elegans, the Bcl-2 homologue CED-9 prevents cell death by antagonizing the death-promoting activities of CED-3, a member of the Caspase family of death proteases, and of CED-4, a protein with no known mammalian homologue. Here we show that **CED-9** interacts physically with CED-4. **Mutations** that reduce or eliminate **CED-9** activity also disrupt its ability to bind CED-4, suggesting that this interaction is important for CED-9 function. Thus, CED-9 might control C. elegans cell death by binding to and regulating CED-4 activity. We propose that mammalian Bcl-2 family members might control apoptosis in a similar way through interaction and regulation of CED-4 homologues or analogues.

L3 ANSWER 8 OF 12 CAPLUS COPYRIGHT 2003 ACS
AN 1997:804305 CAPLUS
DN 128:125084
TI Mutational analysis of the interacting cell death regulators CED-9 and CED-4
AU Ottilie, Sabine; Wang, Yan; Banks, Sean; Chang, Julia; Vigna, Nicole J.; Weeks, Suzanne; Armstrong, Robert C.; Fritz, Lawrence C.; Oltersdorf, Tilman
CS IDUN Pharmaceuticals, Inc., La Jolla, CA, 92037, USA
SO Cell Death and Differentiation (1997), 4(7), 526-533
CODEN: CDDIEK; ISSN: 1350-9047
PB Stockton Press
DT Journal
LA English
AB The genes ced-3, ced-4 and ced-9 are central components in the cell death pathway of the nematode C. elegans. Ced-9, which functions to inhibit cell death, is homologous to the Bcl-2 family of mammalian anti-apoptotic genes. The ced-3 gene encodes a protein homologous to the caspases, a family of cysteine proteases involved in the execution of programmed cell death. It has recently been demonstrated that CED-4, an inducer of apoptosis for which no mammalian equiv. has been reported, can interact with CED-9 and Bcl-xL. Here we confirm that CED-9 and CED-4 interact and using a series of deletion mutants, demonstrate that only short N-terminal deletions are tolerated in each mol. without loss-of-interaction. Two loss-of-function point mutations in different regions of CED-4 also lead to a significant loss of interaction suggesting further that the relevant interaction domains are not short linear sequences, but rather, are formed by more complex structural determinants in each mol. Furthermore, we demonstrate that CED-4 not only interacts with Bcl-xL but also with its homolog, Bcl-2, and that the unstructured loop region present in Bcl-xL and Bcl-2 can regulate the CED-4 interaction. Lastly, we show that a BH3 peptide that can inhibit Bcl-2 family interactions also inhibits the interaction between Bcl-xL and CED-4.

L3 ANSWER 9 OF 12 MEDLINE DUPLICATE 6
AN 94244600 MEDLINE
DN 94244600 PubMed ID: 8187756
TI Baculovirus p35 prevents developmentally programmed cell death and rescues a ced-9 mutant in the nematode Caenorhabditis elegans.
AU Sugimoto A; Friesen P D; Rothman J H
CS Department of Biochemistry, University of Wisconsin-Madison 53706.
NC 1RO1-GM48137 (NIGMS)
SO EMBO JOURNAL, (1994 May 1) 13 (9) 2023-8.
Journal code: 8208664. ISSN: 0261-4189.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English

FS Priority Journals
EM 199406
ED Entered STN: 19940629
Last Updated on STN: 19940629
Entered Medline: 19940617

AB Programmed cell death, or apoptosis, occurs throughout the course of normal development in most animals and can also be elicited by a number of stimuli such as growth factor deprivation and viral infection. Certain morphological and biochemical characteristics of programmed cell death are similar among different tissues and species. During development of the nematode Caenorhabditis elegans, a single genetic pathway promotes the death of selected cells in a lineally fixed pattern. This pathway appears to be conserved among animal species. The baculovirus p35-encoding gene (p35) is an inhibitor of virus-induced apoptosis in insect cells. Here we demonstrate that expression of p35 in C. elegans prevents death of cells normally programmed to die. This suppression of developmentally programmed cell death results in appearance of extra surviving cells. Expression of p35 can rescue the embryonic lethality of a **mutation** in **ced-9**, an endogenous gene homologous to the mammalian apoptotic suppressor bcl-2, whose absence leads to ectopic cell deaths. These results support the hypothesis that viral infection can activate the same cell death pathway as is used during normal development and suggest that baculovirus p35 may act downstream or independently of ced-9 in this pathway.

L3 ANSWER 10 OF 12 MEDLINE DUPLICATE 7
AN 94239527 MEDLINE
DN 94239527 PubMed ID: 7910376
TI Activation of C. elegans cell death protein CED-9 by an amino-acid substitution in a domain conserved in Bcl-2.
CM Comment in: Nature. 1994 May 26;369(6478):272-3
AU Hengartner M O; Horvitz H R
CS Howard Hughes Medical Institute, Department of Biology, Massachusetts Institute of Technology, Cambridge 02139.
SO NATURE, (1994 May 26) 369 (6478) 318-20.
Journal code: 0410462. ISSN: 0028-0836.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199406
ED Entered STN: 19940621
Last Updated on STN: 19950206
Entered Medline: 19940615

AB The Caenorhabditis elegans gene ced-9 and the human proto-oncogene bcl-2, both of which protect cells from programmed cell death, are members of the same gene family. ced-9 and bcl-2 were discovered because of the effects of dominant gain-of-function mutations. Such bcl-2 mutations, which are commonly found in follicular lymphoma, are translocations that result in over-expression of a normal Bcl-2 protein in B cells. Here we report that, by contrast, the **ced-9**(n1950) gain-of-function **mutation** affects the open reading frame of ced-9 and results in a glycine-to-glutamate substitution in a region highly conserved among all ced-9/bcl-2 family members. We conclude that this glycine has an important function in ced-9 regulation, and we suggest that alteration of this glycine in other members of the ced-9/bcl-2 family might lead to oncogenic activation. We also present genetic evidence suggesting that the CED-9 protein might exist in two distinct forms that have opposite effects on cell death.

L3 ANSWER 11 OF 12 CAPLUS COPYRIGHT 2003 ACS
AN 1994:237601 CAPLUS
DN 120:237601
TI A gene which prevents programmed cell death

IN Horvitz, H. Robert; Hengartner, Michael
PA Massachusetts Institute of Technology, USA
SO PCT Int. Appl., 111 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9325683	A1	19931223	WO 1993-US5651	19930614
	W: CA, JP				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	US 6312947	B1	20011106	US 1999-234186	19990120
	US 6465617	B1	20021015	US 1999-233527	19990120
PRAI	US 1992-898933	A	19920612		
	US 1992-927681	A	19920810		
	US 1994-288295	B1	19940810		
	US 1997-801248	A3	19970219		

AB Gene ced-9, a cell death-protective gene from the nematode Caenorhabditis elegans, has been identified, sequenced, and characterized. Ced-9 is essential for C. elegans development and apparently functions by protecting cells which normally live during development from programmed cell death. Mutations which constitutively activate and inactivate the ced-9 gene are also described. Ced-9 was shown to function by antagonizing the activities of the cell death genes, ced-3 and ced-4. The protein product of the human oncogene bcl-2 was found to have a similar sequence to the ced-9 protein. Methods and agents for both increasing and decreasing the occurrence of cell death are described that are potentially useful for diagnosis, prevention and therapy of diseases and conditions involving cell death; for the treatment of viral, parasitic, and other types of infection; and for killing organisms that are detrimental or potentially detrimental to the environment or to humans, pets, livestock, or agriculture.

L3 ANSWER 12 OF 12 MEDLINE DUPLICATE 8
AN 92220162 MEDLINE
DN 92220162 PubMed ID: 1560823
TI Caenorhabditis elegans gene ced-9 protects cells from programmed cell death.
AU Hengartner M O; Ellis R E; Horvitz H R
CS Howard Hughes Medical Institute, Department of Biology, Massachusetts Institute of Technology, Cambridge 02139.
SO NATURE, (1992 Apr 9) 356 (6369) 494-9.
Journal code: 0410462. ISSN: 0028-0836.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199205
ED Entered STN: 19920529
Last Updated on STN: 19920529
Entered Medline: 19920514

AB The gene ced-9 of the nematode Caenorhabditis elegans acts to protect cells from programmed cell death. A **mutation** that abnormally activates **ced-9** prevents the cell deaths that occur during normal C. elegans development. Conversely, **mutations** that inactivate **ced-9** cause cells that normally live to undergo programmed cell death; these mutations result in embryonic lethality, indicating that ced-9 function is essential for development. The ced-9 gene functions by negatively regulating the activities of other genes that are required for the process of programmed cell death.

=>